their data support the contention that sidestream smoke is more genotoxic than mainstream smoke.

Excerpts

"In the present study we have applied MS and SS cigarette smoke condensates topically in equivalent amounts to mice and have determined the relative levels of adducts produced in skin and other tissues. These data, together with results from the treatment of human skin in culture and DNA in vitro, provide evidence for the direct and superior genotoxicity of SS cigarette smoke over that of MS smoke."

"In the five mouse tissues studied both MS-CSC and SS-CSC produced characteristic diagonal radioactive zones . . . indicative of the formation of multiple DNA adducts. In three tissues (skin, lung and kidney), SS-CSC induced greater total adduct levels than MS-CSC (statistically significant in skin and kidney). However, greater adduct levels induced by MS-CSC were recorded for heart and bladder DNA (not statistically significant). Similar results to those found in mouse skin were obtained with human skin; SS-CSC induced [an approximately] 2-fold greater level of DNA adducts than MS-CSC. Incubation of DNA directly with condensates in vitro demonstrated that DNA adducts could be formed without an exogenous metabolizing system. This direct interaction of condensates with DNA occurred at similar levels for both MS- and SS-CSC, although inclusion of an oxygen radical-generating system enhanced the SS-CSC binding to a greater extent than that of the MS-CSC."

"The poorer efficiency of combustion by which SS smoke is generated results in the formation of greater levels of numerous carcinogenic components than are present in actively inhaled MS smoke. . . . In this report we have demonstrated that the increased levels of carcinogens in SS smoke are translated into a greater adduct-forming potential in certain mouse tissues and in human skin. In addition, the smoke condensates were shown to contain components that were direct-acting in their ability to form DNA adducts, a property which may, in part, be due to the presence of the active oxygen species known to be present in cigarette smoke. . . . Such reactive species may themselves generate DNA adducts or they may activate other genotoxic components of the CSCs; the inclusion of an oxygen free-radical-generating system was shown to enhance the direct adduct formation of both MS- and SS-CSC, the latter being enhanced to a greater extent. The relative contributions of direct-acting and metabolically-activated components to the overall DNA binding in vivo remains to be determined."

"Thus, the data presented in this paper indicate that, on a unit to unit basis, SS cigarette smoke appears more genotoxic than MS smoke and that, although the SS component of environmental tobacco smoke may be diluted considerably in air, the potentially harmful effects of passive smoking are demonstrable at a molecular level."

[2] **Letters to the Editor Regarding Eliopoulos, C., Klein, J., Phan, M.K., Knie, B., Greenwald, M., Chitayat, D., and Koren, G., "Hair Concentrations of Nicotine and Cotinine in Women and Their Newborn Infants," *Journal of the American Medical Association* 271(8): 621-623, 1994 [Issue 76, Item 40]**

Letters to the editor were recently published concerning this paper, which reported measurements of nicotine and cotinine from the hair of newborns whose mothers smoked and whose mothers were reportedly exposed to ETS. *See* issue 67 of this Report, March 4, 1994. A letter by Joseph C. Avakoff, M.D., was published, as was a reply by one of the authors of the original paper, Gideon Koren. The letters appear at *Journal of the American Medical Association* 271(24): 1904-1905, 1994.

In his letter, Avakoff notes that Eliopoulos, et al., had presented data indicating a level of 0.4 ng/mg of nicotine in the hair of infants from nonsmoking mothers, and a level of 0.28 ng/mg for infants whose mothers were reportedly exposed to ETS. He questions the source of the nicotine in the nonsmokers, and also questions why the nonsmokers had a higher level than did those reportedly exposed to ETS.

Koren's reply states that the difference in nicotine levels was not statistically significant, while the difference in cotinine levels was. He proposes that cotinine is a more accurate marker of exposure. Koren then notes that "[l]ow exposure to nicotine is part of most people's diet," although he claims that "even in cases of 'true' nonsmokers, none of us can escape all the nicotine that surrounds us."

[3] **Frank, J.E., Johnson, H., Flanagan, V., and Hoffman, D., "Maternal Passive Smoking: A Potential Fetal Risk," *Pediatric Research* 35 (4 Part 2): 269A, 1994 [Issue 78, Item 33]**

In this meeting abstract, the authors report on cotinine levels detected in samples of fetal meconium (fecal matter). They claim that cotinine may be "a marker of potential risk."

EXCERPTS:

"Maternal smoking is an accepted risk factor for the fetus. Involuntary or passive smoking is considered a health hazard. We designed a study to determine the presence of nicotine metabolite (cotinine) in meconium of newborns of mothers who were exposed to passive smoke. . . . We defined passive smoking as a non-smoker exposed to > or = to one hour/week of smoke at the workplace or home."

"Results . . . indicate that cotinine concentration >20 ng/ml was detected in most passive and active smoking mothers. One passive smoker and one active smoker were <20 ng/ml. One self-reported, non-exposed, non-smoker was in the intermediate level associated with passive smoking, and two were in the high level associated with active smoking. This may indicate inaccurate reporting of smoking history or exposure. More importantly, it may suggest that non-smoking mothers who think they are not exposed to smoke are unaware of the smoke in their surrounding environment. Cotinine in meconium of infants born to mothers passively exposed to smoke represents a marker of potential risk."

[4] Guerin, M.R., "Environmental Tobacco Smoke Exposure Assessment." In: *Environmental Tobacco Smoke.* H. Kasuga (ed.). Springer-Verlag, 4-27, 1993 [Issue 81, Item 22]

In this review, the author concludes that there is not currently a "completely satisfactory method" for assessing ETS exposure. He also notes that area and personal monitoring studies report low levels of ETS constituents in most indoor environments.

EXCERPTS:

"This paper discusses approaches to exposure assessment and summarizes data on indoor air concentrations of ETS constituents."

"Environmental tobacco smoke is believed to contain many of the same constituents as mainstream smoke but in different proportions. . . . Sidestream to mainstream ratios vary widely by chemical class and by individual chemicals within classes, resulting in a different quantitative composition of ETS as compared to mainstream smoke. Other major differences are the result of dilution in ambient air."

"The physical and chemical composition of ETS varies with time and in space depending on ventilation, indoor surfaces, temperature, humidity, and background contaminants."

"An important but often overlooked property of tobacco smoke is that it is readily detectable by sight and smell. Observing smoking in the vicinity of a subject can be construed as exposure to ETS regardless of its distance form the subject or the time spent by the subject in the area. Detecting the odor of tobacco smoke can be construed as exposure to ETS even though the odor is due to long-past smoking."

"The use of sidestream/mainstream ratios is now known to be a poor means of estimating generation rates."

"The concept of cigarette equivalents continues to be used. The principal difficulty is that the results are highly dependent on the chemical chosen to measure cigarette equivalents."

"The accuracy of results obtained using surrogates (e.g., spousal smoking) and questionnaires (e.g., self-assessment of exposure and time/activity patterns) have long been questioned, but it appears that these approaches are at least useful for gross categorization."

"It is generally agreed that the results of questionnaires should be supplemented with results of air monitoring or biomarker measurements whenever possible. This is necessary to reduce misclassification of subjects and to better define the levels of exposure of actual concern."

"Concentrations of representative ETS constituents in commonly encountered indoor environments are summarized . . . and concentration differences between smoking and nonsmoking environments for several constituents are given . . . With the exception of ETS-unique or ETS-predominant constituents (e.g., nicotine and N-nitrosamines, respectively), concentrations in indoor air are similar. In all cases, however, indoor air concentrations are higher than those outdoors, and concentrations in smoking environments tend to exceed those in non-smoking environments."

"An important observation from area sampling studies is that air concentrations are highly skewed toward the low end of the concentration range, i.e., most environments sampled are minimally contaminated."

"The results suggest, however, that low level exposure is more commonly encountered than is high level exposure."

"A wide range of ETS constituent concentrations are [sic] found in commonly encountered environments. Exposure is largely determined by the time spent in each environment. It is for this reason that personal sampling is gaining favor over area sampling for exposure assessment."

"No completely satisfactory method for measuring exposure to ETS currently exists. Questionnaires to estimate ETS exposure are only now being validated. Biomarker measurements suffer from a poor understanding of the relationship of the marker chemical to ETS as a whole and from intersubject variability. Area air sampling provides a measure of potential exposure but requires a measure of exposure time to determine actual exposure. Personal air sampling is only beginning to be deployed. Both area and personal sampling share the limitation of biomarker measurements -- the relationship between the individual ETS constituents is variable and poorly understood."

"Both area and personal monitoring studies have tended to indicate that air concentrations of ETS constituents are skewed towards the low end of the concentration range, indicating rather minimal contamination of most indoor air spaces from smoking. Yet circumstances have been identified with clearly measurable levels of exposure (e.g., bars and nightclubs), particularly in the absence of adequate ventilation. Over time, definitive measures of ETS exposure need to be developed to test current hypotheses concerning possible ETS-related health effects and, if validated, to determine exposure levels of concern."

[5] Hernandez, A., Daffonchio, L., Brandolini, L., and Zuccari, G., "Effect of a Mucoactive Compound (CO 1408) on Airway Hyperreactivity and Inflammation Induced by Passive Cigarette Smoke Exposure in Guinea-Pigs," *European Respiratory Journal* 7: 693-697, 1994 [Issue 81, Item 23]

According to the authors of these studies, ETS exposure of guinea pigs produced histologically confirmed inflammation of the lungs and was also associated with airway hyperreactivity, measured as contractions in isolated lung tissue.

EXCERPTS:

"Environmental exposure to tobacco smoke contributes to the onset of several lung diseases, e.g. chronic bronchitis and asthma, including an increase in airway reactivity. We have investigated the effect of a new mucoactive compound, CO 1408, on airway hyperreactivity and lung inflammation induced in guinea-pigs by passive cigarette smoke exposure."

"Animals were exposed to cigarette smoke in a Plexi-glass box, three times a day for four days. Airway reactivity to histamine was assessed *ex-vivo* in lung parenchymal strips. As a measure of lung inflammation, the number of leucocytes was evaluated in bronchoalveolar lavage (BAL) fluids and histological sections."

"CO 1408 itself did not modify the histamine induced contractions."

"Passive cigarette smoke exposure in conscious guinea-pigs resulted in airway hyperreactivity, shown by an increase in histamine induced contractions in isolated parenchymal strips."

"At a dose of 400 mg/kg *per os*, CO 1408 completely inhibited the cigarette smoke induced increased reactivity to histamine."

"There was a significant increase in total cell count, eosinophils and macrophages, in the BAL fluids obtained from smoke-exposed animals, when compared to sham-exposed controls. CO 1408 . . . inhibited the changes in number of inflammatory cells in animals exposed to smoke."

"The results obtained from histological analysis confirmed the development of an inflammatory reaction within the lung after passive cigarette smoke exposure."

"CO 1408 is a new mucoactive drug with potential effectiveness in some obstructive lung diseases . . . Our results demonstrate that this compound also possesses an inhibitory effect against the smoke-induced airway hyperreactivity and inflammation, which was dose-dependent only at the highest dose tested."

"These data suggest that recruitment of inflammatory cells into the airway lumen could be more relevant to the induction of the airway hyperreactivity to histamine than recruitment in lung parenchyma."

[6] Mukherjee, S., Nayyar, T., Chytil, F., and Das, S.K., "Mainstream and Sidestream Cigarette Smoke Exposure Increases Retinol in Guinea Pig Lungs," *The FASEB Journal* 8(7): A1463, 1994 [Issue 80, Item 38]

In this abstract, the authors report that guinea pigs exposed to either mainstream or sidestream smoke exhibited elevated retinol levels in the lungs and unspecified "deleterious" changes in lung structure.

EXCERPTS:

"We have studied in guinea pigs the effects of cigarette smoke exposure on retinol levels in plasma, lung, lung lavage and liver. . . . Three weeks old male guinea pigs were exposed to mainstream (MS), sidestream (SS) or sham smoke, generated twice daily from 3 cigarettes for 6 weeks. . . . After 6 weeks of smoking the plasma retinol

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levels were lower in both smoke exposed groups whereas the lung retinol levels were 7.6- and 8.3-fold higher in MS and SS groups, respectively when compared to the levels in the sham group. After the 6-week recovery period plasma retinol of both MS and SS groups reached the control levels whereas no such effect was noted on the lung retinol level. Electronmicroscopy of the lungs showed [deleterious] alterations in the morphology of the lungs in both MS and SS groups. Although the mechanism(s) involved in the elevation of retinol content of the lung due to smoke exposure remains to be elucidated, it is of interest that elevation of retinol content and alteration of lung morphology occurred not only in the MS group but also in the SS group."

[7] **Ogborn, C.J., Duggan, A.K., and DeAngelis, C., "Urinary Cotinine as a Measure of Passive Smoke Exposure in Asthmatic Children," *Clinical Pediatrics* 33(4): 220-226, 1994 [Issue 76, Item 41]**

Using cotinine measurements and parental questionnaires, the authors of this paper compared reported ETS exposures at the time of acute asthmatic symptoms and at a symptom-free time. They reported no significant difference in exposure between the asthmatic and the "well" visit.

EXCERPTS:

"The goals of this study were to determine (1) the association between level of passive smoke exposure and acute symptoms in asthmatic children, and (2) the association between objective measures of exposure (urinary cotinine and CCR [cotinine to creatinine ratio]) and parental report of exposure in these children."

"[T]here was no significant difference between objective measures of smoke exposure at the acute and well visits. Mean urinary cotinine levels were 81 ± 62 ng/mL at the acute visit and 77 ± 57 ng/mL at the well visit."

"Parental report of amount of smoke exposure, measured four ways using questionnaire data, generally showed no significant difference in exposure rate or intensity between the acute and well visits. . . . The exception to this is the graded question which was phrased as follows: 'How much smoke has your child been exposed to in the last 48 hours?' ('None, a little, some, a lot, uncertain'). For this question there was a difference between the acute and the well visits that was statistically significant."

"At both the acute and well visits, parental responses were positively associated with urinary measures of exposure. In most instances, the level of association was statistically significant, and for the others it approached statistical significance."

"The 77% household smoke exposure rate in our study population was disturbingly high."

"We initially thought we might find some children who had large fluctuations in their smoke exposure from time to time and that this might correlate with their asthma attacks. We found no significant difference in objective measures of passive smoke exposure between one visit and the next. Additionally, we found that neither the mean number of cigarettes per day nor hours per day of exposure changed from one visit to the next."

"Our inability to demonstrate a significant difference in measures of smoke exposure from the acute to the well visit is most likely because there was no difference."

"We are left with the conclusion that this particular study population of asthmatic children has a high exposure level to environmental tobacco smoke and that this level of exposure is relatively constant from time to time. . . . Thus, it seems plausible that among asthmatic children with chronic exposure, their asthma attacks may not be triggered by discrete increases in passive cigarette smoke exposure. We cannot determine whether asthmatic children who are not usually exposed to cigarette smoke might have an asthma attack triggered by acute exposure to smoke, since we had so few children in this category."

"However, we do not conclude that cigarette smoke exposure has no influence on symptoms of asthma in children. Rather, these children belong to a group whose chronic respiratory illness, asthma, might have been engendered by exposure to smoke in the first place. The irritant effect of the intense passive smoke exposure might create and maintain in the lungs an inflammatory process which otherwise would not exist."

[8] **Teredesai, A., and Pruehs, D., "Histopathological Findings in the Rat and Hamster Respiratory Tract in a 90-Day Inhalation Study Using Fresh Sidestream Smoke of the Standard Reference Cigarette 2R1." In: *Toxic and Carcinogenic Effects of Solid Particles in the Respiratory Tract.* U. Mohr, D.L. Dungworth, J.L. Mauderly, and G. Oberdorster (eds.). Washington, ILSI Press, 629-635, 1994 [Issue 80, Item 39]**

The authors of this paper report that, in rats and hamsters exposed to sidestream smoke at levels higher than those normally associated with "real-world" conditions, minor, reversible histopathological changes were

observed in the larynx and vocal cords of rats. No such changes were reported in hamsters.

EXCERPTS:

"While histopathological changes in the respiratory tract of rats and hamsters in short-term and long-term inhalation studies with mainstream smoke (MS) have been reported extensively in literature, there are only three published inhalation studies on rats and on rats and hamsters with sidestream smoke in which histopathology is the main end point."

"The animals were nose-only-exposed to fresh sidestream smoke (FSS) for 7 hours/day, 7 days/week for 90 days . . . The TPM concentrations in the FSS of 2 and 6 ug/L are significantly above the levels reached in occupied spaces with smoking."

"Twenty male rats and 20 male hamsters per group were allocated to one sham exposure group and two FSS groups. Of these, 10 rats and 10 hamsters per group were kept for a 21-day postinhalation period."

"In both species, no smoke-exposure-related effects were seen for in-life observations, mortality, body weight, organ weights, and gross pathology."

"The severity of these findings in rats was slight, and they were observed mainly in the high FSS concentration group. No smoke-exposure-related histopathological changes were observed in trachea and lungs. A dose-dependent increase in epithelial thickness in the larynx compared to sham was observed at the floor of the larynx and at the lower medial surface of the vocal cords . . . All the aforementioned findings reversed completely during the 21-day postinhalation period, the exception being hyperplasia at the vocal cords, which was still present in three rats of the high FSS concentration group. All findings are considered to be an adaptive response to repeated irritation."

"In the hamster respiratory tract, no histopathological changes were seen."

"The reserve cell hyperplasia of the rat nasal respiratory epithelium and the lack of findings for the hamster are in accordance with published literature. The slight hyperplasia and the slight squamous metaplasia found in the rat laryngeal epithelium have not been reported to date in the literature. The changes were reversible and are considered to be an adaptive response to repeated irritation."

"The No Observed Effect Level (NOEL) for all FSS-related findings for this study is between 2 and 6 ug TPM/L for rats. This concentration range is between 1 and 2 orders of magnitude above the average environmental concentration."

**[9] Witschi, H., Lundgaard, S.M., Rajini, P., Hendrickx, A.G., and Last, J.A., "Effects of Exposure to Nicotine and to Sidestream Smoke on Pregnancy Outcome in Rats," *Toxicology Letters* 71: 279-286, 1994 [Issue 79, Item 35]**

In this study, the effects of exposure to nicotine and sidestream smoke (SS) on pregnant rats were examined. The authors report that continuous exposure to nicotine resulted in a reduction in the number of pregnancies carried to term and that in rats exposed to SS (a surrogate for ETS), a decrease in litter size and implantation sites was observed.

EXCERPTS:

"Several agents have been suggested as playing a causative role in the development of intrauterine growth retardation. Maternal undernutrition caused by smoking, CO induced fetal hypoxia, or the pharmacologic effects of nicotine which can cause epinephrine release with resulting constriction of the uteroplacental vessels have all been considered as key mechanisms in intrauterine growth retardation by tobacco."

"Recently a new nicotine delivery system became available. Patches that deliver nicotine transdermally have been introduced as an aid for smoking cessation. The patches also allow an investigator to expose animals continuously to defined doses of nicotine. . . . We investigated whether continuous infusion of nicotine via the transdermal route would adversely affect fetal development in rats."

"In a separate experiment we examined the effects of a complex airborne mixture containing nicotine on pregnancy outcome. Pregnant rats were exposed to sidestream smoke (SS)."

"Some of the animals were exposed to the nicotine during the entire pregnancy (days 2 through 19), whereas other groups were exposed to nicotine only during the first trimester (days 2 through 7) of the gestation period."

"In unhandled control animals, there was a 100% incidence of pregnancies. . . . Exposure to nicotine greatly reduced the number of pregnancies that were carried successfully to term. When nicotine was delivered over the full duration of gestation, none of the animals exposed to 3.5 mg of nicotine per day remained pregnant. In the group of animals exposed throughout gestation to 1.75 mg of nicotine a day, fewer than half of the animals remained pregnant. . . . Exposure to nicotine only during

the first trimester of pregnancy had a less dramatic effect and pregnancy failure was statistically significant only in animals exposed to 3.5 mg of nicotine per day."

"[T]he animals exposed to [sidestream] smoke appeared to have gained somewhat less weight, although the difference to the controls was statistically not significant. . . . [I]t was found that the average number of implantations and the number of live pups per litter was lower in the animals that had been exposed to sidestream smoke during the first half of pregnancy."

"The data presented in this paper show that continuous exposure of rats to nicotine throughout pregnancy results in a significant reduction in the number of pregnancies that are carried to term. Levels of plasma nicotine associated with clear cut pregnancy failure ranged on average from 43 to 241 ng/ml. In addition, this pregnancy loss effect also seemed to occur in a dose-dependent manner when nicotine was given continuously through the first trimester of gestation or, at the highest dose used, even through days 2 to 5 of pregnancy. This suggests that nicotine may have a preimplantation effect, a possibility that will have to be explored further. On the other hand, it was not possible to document an effect of nicotine administration on birth weight."

"Exposure of pregnant rats to SS, a surrogate for environmental tobacco smoke, produced plasma nicotine levels that were about 15 to 25% of those found in animals that had received 1.75 mg of nicotine per day. There were close to peak plasma concentrations of nicotine typically seen in humans carrying transdermal nicotine delivery systems. In humans there are data that suggest that ETS adversely affects fetal development. Under the conditions of the present experiment, a significant decrease in litter size and a reduction in implantation sites was observed. However, in view of the multiple other components in tobacco smoke that may adversely affect pregnancy outcome, most notably CO, the influence of SS on pregnancy needs further detailed evaluation."

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## INDOOR AIR QUALITY

[1] **Brown, S.K., Sim, M.R., Abramson, M.J., and Gray, C.N., "Concentrations of Volatile Organic Compounds in Indoor Air -- A Review,"** *Indoor Air* **4: 123-134, 1994 [Issue 81, Item 24]**

This review of published literature concludes that VOC levels in dwellings are generally higher than those in public buildings, and that VOC levels in new buildings are greater than those in older buildings. The authors also suggest that any relationship between VOC levels and building-related complaints was inconclusive.

EXCERPTS:

"A review is presented of investigations of volatile organic compound (VOC) concentrations in indoor air of buildings of different classifications (dwellings, offices, schools, hospitals) and categories (established, new and complaint buildings). Measured concentrations obtained from the published literature and from research in progress overseas were pooled so that VOC concentration profiles could be derived for each building classification/category."

"[C]oncentrations could be derived for only 80 compounds in established dwellings. Nineteen of these VOCs exhibited . . . concentrations above 5 ug/$m^3$ and these are considered to be predominant indoor air compounds. Only one (ethanol) exceeded 50 ug/$m^3$."

"Comparative . . . concentrations in dwellings and public buildings could be derived for 36 compounds. For most of these compounds, it was found that concentrations in dwellings were greater (to a 5% statistical significance) than those in public buildings, typically by a factor of 2 or more. . . . This was also found for TVOC results."

"[C]oncentrations in new buildings were considerably greater than in established buildings, often by an order of magnitude or more."

"[I]ndoor concentrations were significantly elevated above those outdoors for virtually all compounds, indicating that they were emitted from indoor rather than outdoor sources. This is consistent with the suggestion that inhalation of indoor air rather than outdoor air is a more significant factor for environmental exposure to VOCs."

"It is not known why VOC and TVOC concentrations derived for dwellings are greater than those for public buildings. Many of the dwelling measurements were made in the US TEAM studies and the greater concentrations may reflect low building ventilation levels or an influence of the use of personal sampling in TEAM studies of dwellings in comparison to static sampling used in most other studies as discussed earlier."

"No conclusions can be drawn about VOC and TVOC concentrations in complaint buildings relative to those in established buildings. The number of complaint buildings investigated was small, the nature and

incidence of occupant complaints received little attention, and TVOC concentrations were measured using different definitions of unknown significance to occupant exposure and health."

"By pooling the results of a large number of surveys in different countries it has been found that:"

"(a) the mean concentration of each VOC in established buildings is generally below 50 ug/m$^3$, with most below 5 ug/m$^3$, while TVOC concentrations are substantially higher (e.g. 1100 ug/m$^3$ in dwellings), reflecting the large number of compounds present;"

"(b) the mean VOC and TVOC concentrations in established dwellings are generally greater than those in established public buildings, for unknown reasons;"

"(c) VOC concentrations in new buildings are much greater than those in established buildings, often by an order of magnitude or more, and appear to arise from construction materials and building contents, the VOC emission characteristics of which can be measured for source control; and"

"(d) VOC and TVOC concentrations in complaint buildings have been measured to a limited extent and may or may not be greater than those in established buildings."

[2] Junge, B., "Passive Smoking Aboard Passenger Aircraft," *Tobacco Control* 3: 50-58, 1994 [Issue 77, Item 40]

Based on a review of the literature on ETS measurements on aircraft, the author concludes that ETS exposures are high, even in nonsmoking areas, and claims that children, pregnant women, "susceptible" individuals, and cabin staff are at risk from such exposures.

EXCERPTS:

"A review of the relevant literature on measurements of tobacco smoke constituents in aircraft and other closed environments reveals that being seated in the non-smoking section of an aircraft does not provide effective protection against the involuntary inhalation of tobacco smoke. In certain instances, smoke concentrations higher than the average level in the smoking section may be present. Particularly in the borderline area between the smoking and non-smoking sections (ie, one to three rows in front of or behind a smoking section), levels may be equal to or above those in the smoking section. This is particularly the case where ventilation makes partial use of recirculated air."

"Pollutant concentrations from tobacco smoke aboard aircraft in smoking sections and the bordering non-smoking sections in part clearly exceed the levels in other indoor situations where smoking takes place. Levels in the non-smoking sections of aircraft (beyond the areas bordering smoking sections) are similar to those in homes where smoking takes place. In the majority of studies, health risks associated with passive smoking have been found in persons living in smokers' households."

"Although exposure of the average air passenger lasts only for a comparatively short period, damage to health from such passive smoking should be principally the same, although clearly of a lesser impact, as in persons continuously exposed to tobacco smoke at home or at the workplace. The concentrations measured may cause an impairment of well-being and an elevated health risk for children, pregnant women, persons with a high susceptibility to tobacco smoke, frequent flyers, and especially cabin staff."

"Being seated in the non-smoking section of an aircraft cabin does not provide adequate protection against involuntary inhalation of tobacco smoke. Even at some distance from the smoking section, single concentrations may reach levels similar to average levels in the smoking section."

"Lower nicotine and RSP values were measured in certain types of aircraft (eg, B 747) or in some non-smoking sections in the front part of the cabin (first class, business class). These levels varied, however, depending on the number of cigarettes consumed in the respective smoking sections. Were ventilation truly effective, it should result in consistently low levels irrespective of the number of cigarettes smoked."

"In some of the studies reviewed, tobacco-related parameters were measured in blood and/or urine, demonstrating that tobacco smoke is not just present in cabin air but is, in fact, inhaled by passengers and flight attendants. Tobacco smoke components have been detected in body fluids and have been found to correlate with tobacco smoke levels in cabin air."

"The risk of *chronic* respiratory disease or lung cancer from passive smoking aboard aircraft is low for *average* passengers given their normally short exposure period. However, this exposure should still be eliminated because it contributes to their total exposure to ETS, a documented human carcinogen."

"Acute effects of tobacco smoke exposure in the non-smoking section are more likely to occur, even after short-term exposure. In particular, the concentrations

measured in the two or three rows adjacent to the smoking section may produce irritation of the eyes and the respiratory tract and discomfort. Children, pregnant women and persons highly sensitive to tobacco smoke should avoid such exposure. Both acute and chronic exposure to cigarette smoke produce a variety of health effects."

"Numerous airlines operating abroad and no less than three (smaller companies) operating in Germany have resolved the problem by introducing non-smoking flights. This is the best and least expensive means of protecting non-smokers from passive smoking."

[3] **Leaderer, B.P., Koutrakis, P., Briggs, S.L.K., and Rizzuto, J., "The Mass Concentration and Elemental Composition of Indoor Aerosols in Suffolk and Onondaga Counties, New York,"** ***Indoor Air*** **4: 23-34, 1994 [Issue 80, Item 40]**

Reporting on analyses of aerosol samples collected within and outside nearly 400 homes in New York State, the authors claim that smoking was a more important source of aerosols than were various forms of heating, including wood burning stoves.

EXCERPTS:

"Indoor and outdoor aerosol sampling was conducted in two New York State Counties, Suffolk and Onondaga, during the period of January 6 and April 15, 1986. Week-long fine particle mass samples were collected indoors and outdoors for a total of 596 samples taken in 394 homes."

"Homes included in this study had one or more of the following sources: cigarette smoking, kerosene heaters, wood burning and gas stoves. Homes with none of the above sources were also included."

"The results of these analyses showed that gas stoves and humidifiers do not contribute to indoor aerosol mass and elemental concentrations."

"Although the above preliminary description of the indoor and outdoor data does not allow for a profound understanding of the origin of the indoor aerosols, it gave us enough information about the signature and relative importance of the different indoor aerosol sources. For instance, smoking appears to be the most important source among the five source types examined for this study. To a lesser extent kerosene heaters and wood burning can affect indoor aerosol concentrations. Furthermore, the results of the comparison of aerosol concentrations between different rooms, kitchen or other living areas versus living area, showed no room-to-room variation in aerosol mass and elemental concentrations."

"The main objectives of this paper were (a) presentation of the study design; (b) description of the sampling and analysis techniques; (c) reclassification of the collected data to minimize home groups; (d) comparison of the mean outdoor and indoor aerosol mass and elemental concentrations; and (e) examination of room-to-room variations in the aerosol concentrations."

[4] **Patuszka, J.S., Gorny, R., and Sokal, J., "Influence of Tobacco Smoking on the Particulate Mass Size Distribution Indoors in Upper Silesia, Poland,"** ***Zentralblatt Hyg Umweltmed*** **195: 190, 1994 [Issue 77, Item 41]**

This meeting abstract suggests that outdoor pollution, tobacco smoking, and coal burning are the main contributors to particulates in indoor air in one region of Poland.

EXCERPTS:

"The residents of the Upper Silesia are acutely aware of the environmental imbalance influencing . . . human health directly as well as indirectly, via the indoor air. . . . [I]ndoor air may be contaminated with airborne particles either penetrating from outdoors or produced indoors. Assessment of exposure to indoor air particulate pollution should also include environmental tobacco smoking. Poland is a country of high tobacco consumption. The magnitude of the problem needs due attention . . . Estimation of the contribution of smoking to indoor air pollution is necessary to assist in implementation of appropriate preventive measures."

"This work contains the preliminary results of the mass size distribution measurements of indoor and outdoor aerosols in homes located in five towns in Upper Silesia. . . . Analysis of the obtained results indicated cigarette smoking and heavy polluted outdoor air as the main sources of particulate matter indoors in Upper Silesia. However the coal combustion in stoves also gives a substantial contribution to indoor levels of particles."

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## SMOKING POLICIES AND RELATED ISSUES

[1] **Brigham, J., Gross, J., Stitzer, M.L., and Felch, L.J., "Effects of a Restricted Work-Site Smoking Policy on Employees Who Smoke,"** ***American Journal of Public Health*** **84: 773-778, 1994 [Issue 80, Item 41]**

The authors of this paper, which reports on a smoking ban in a Maryland hospital, claim that smokers reduced

their cigarette consumption following institution of the ban. In addition, smokers apparently reported some declines in productivity and in perceived well-being when the ban was in effect.

EXCERPTS:

"Among the hospitals joining the smoke-free movement recently was Francis Scott Key Medical Center, part of the Johns Hopkins medical institutions in Baltimore, Md. This institution implemented a hospital-wide smoking ban on July 1, 1989. We used this event as an opportunity to examine the biological, subjective, and behavioral impact of a smoking policy restriction on individual smokers, including objective measures of tobacco smoke exposure. We compared these effects with those in a control group of smokers whose work-site smoking policy remained unrestricted."

"Based on the self-reported smoking amount, consumption of cigarettes during work hours by subjects in the restricted group dropped significantly as a function of the ban."

"Restricted subjects reported an average of 5.89 cigarettes per day at the workstation before the an compared with 0.69 cigarettes after the ban. . . . Before the ban, the restricted group smoked an average of 0.83 cigarettes per day outside the building compared with 2.51 cigarettes after the ban."

"[D]ata revealed that the number of cigarettes smoked per day during off-work hours did not change significantly in either group as a function of the ban."

"[R]estricted group members noted small but significant decreases on self-reports of concentration, productivity, relations with coworkers, and overall well-being. Control subjects showed no changes over time on the work-productivity indices. Only the overall well-being item had a significant between-group difference after the ban, with the restricted group reporting a lower score."

"The most striking finding was that smokers did not compensate for the pack per week reduction either by increased smoking outside of work or by more intensive smoking. Thus, a reduction in overall exposure might be expected."

"Smokers undergoing an abrupt worksite smoking ban showed statistically significant increases in four of the most reliably reported tobacco withdrawal symptoms: difficulty concentrating, craving cigarettes, increased eating, and depression. . . . Restricted smokers reported some difficulties with work productivity and a decline in general well-being."

[2] **Glantz, S.A., and Smith, L.R.A., "The Effect of Ordinances Requiring Smoke-Free Restaurants on Restaurant Sales," *American Journal of Public Health* 84: 1081-1085, 1994 [Issue 81, Item 25]**

Using data on restaurant sales for 30 California and Colorado communities, the authors report that restaurant sales were not adversely affected by smoke-free restaurant ordinances.

EXCERPTS:

"This study analyzes sales tax data for the first 15 US cities to enact smoke-free ordinances affecting restaurants. The California cities of Auburn, Bellflower (which repealed its ordinance in March 1992), Beverly Hills (which amended its ordinance 4 months after it went into force), El Cerrito, Lodi, Martinez, Palo Alto, Paradise, Roseville, Ross, Sacramento, and San Luis Obispo, and the Colorado cities of Aspen, Snowmass Village, and Telluride have had such 100% smoke-free restaurant ordinances in force long enough to assess their effects. We also examined sales tax data from 15 comparison cities similar to the smoke-free cities in population, income, smoking prevalence, and other factors. An analysis of restaurant sales as a fraction of total retail sales, and of restaurant sales in cities with smoke-free restaurant ordinances compared with those in similar cities that do not have smoke-free ordinances, shows no significant effects on business."

"Data were obtained from the California State Board of Equalization and Colorado State Department of Revenue on taxable restaurant sales from 1986 (1982 for Aspen) through 1993 for all 15 cities where ordinances were in force, as well as for 15 similar control communities without smoke-free ordinances during this period. These data were analyzed using multiple regression, including time and a dummy variable for whether an ordinance was in force. Total restaurant sales were analyzed as a fraction of total retail sales and restaurant sales in smoke-free cities vs the comparison cities similar in population, median income, and other factors."

"Smoke-free ordinances generally had no statistically significant effect on the fraction of retail sales that went to restaurants or on total restaurant sales in cities with ordinances compared with those in cities without smoke-free ordinances. There is marginal evidence that the fraction of total retail sales to restaurants increased in two cities . . . and decreased in one city. In a comparison of restaurant sales in one city with an ordinance versus one city without an ordinance, sales increased in one city and decreased in another. The lack of consistent response suggests that these results may simply reflect random variation."

"Beverly Hills is a particularly important case because it has been used by the tobacco industry to support the claim that smoke-free restaurant ordinances are associated with a 30% drop in business. However, data reveal that no such drop in sales occurred upon enactment, and that no increase in sales followed repeal 4 months later. Likewise, despite the fact that the Bellflower ordinance was repealed because of claims that business dropped, the ordinance was actually associated with a marginally significant increase in business."

"This is the first comprehensive study that examines taxable sales data to determine the economic impact of smoke-free restaurant ordinances on restaurant sales."

"The fact that there were no adverse effects on business in these communities supports the conclusion that the results generalize broadly. Further, these 15 cities represent every city that has passed smoke-free ordinances that have been in effect long enough to study."

"A common concern is raised about the possibility that patrons will dine in adjacent communities without such restrictions. Our data address this concern because the cities examined in this study are not isolated communities. . . . If people were leaving these cities to dine in neighboring cities, our analysis would have detected it."

"Had the ordinances affected sales negatively, we would have expected to see an increase in sales following repeal. However, there was no increase in Beverly Hills, and sales dropped in Bellflower after the ordinance was repealed. Thus, legislators and government officials can enact such health and safety requirements to protect patrons and employees in restaurants from the toxins in second-hand tobacco smoke without the fear of adverse economic consequences."

[3] Kinne, S., Kristal, A.R., White, E., and Hunt, J., "Work-Site Smoking Policies: Their Population Impact in Washington State," *American Journal of Public Health* 83: 1031-1033, 1994 [Issue 81, Item 26]

A population-based telephone survey was used in this study, which reports that 81 percent of employed men and 91 percent of employed women in the state of Washington have a smoking policy in their workplace.

EXCERPTS:

"[W]e report data from a population-based telephone survey of Washington State adults describing employed persons' reports of their smoking habits and the content and impact of smoking restrictions in their work sites."

"Eighty-one percent of employed men and 91% of employed women in Washington reported a smoking policy at their place of work."

"In our study, women were subject to more restrictive policies. Thirty-two percent of men and 52% of women worked in settings in which smoking was prohibited. Those in the smallest work sites were most likely to work without a policy. Female and male professionals and female sales and clerical workers were more likely to work under no-smoking conditions. For men, residence in an urbanized area increased the chance of working in a no-smoking site, reflecting the adoption of smoking bans by many governments and private employers in the Puget Sound area."

"Men and women in no-smoking work sites were less likely to be current smokers, and male smokers in no-smoking and restricted sites reported smoking less both at work and elsewhere than did those in companies without polices. This pattern is consistent with three conditions: a work-site policy may affect smoking, the policy may influence where smokers are hired, and sites with nonsmokers may be more likely to adopt restrictions. . . . We found no relation between policy restrictiveness and quitting smoking."

"[W]e found that for men in work sites with any policy, current smokers were more likely to have been hired before and less likely to have been hired after policy adoption than were nonsmokers. This suggests that selection on the basis of smoking status may influence where a man works."

"These findings show that the great majority of employed Washingtonians are subject to restrictive smoking policies of the type that typically reduce exposure to environmental tobacco smoke. These policies may also reduce smoking among the smokers they affect."

[4] Levy, J., "Workplace Smoking: Employers Face New Challenges," *American Journal of Orthodontics and Dentofacial Orthopedics*, (February): 211-212, 1994 [Issue 79, Item 36]

This article discusses regulations and ordinances that employers are faced with today when attempting to accommodate the needs of smokers and nonsmokers in the workplace. According to the author, smoking policies should consider both smokers and nonsmokers, and should be tailored to individual workplaces.

EXCERPTS:

"Although no federal workplace smoking law yet exists, employers face a myriad of state and especially local laws,

regulations and ordinances on smoking -- with the promise of federal regulations to come. Today's employers must carefully develop, implement and enforce workplace smoking policies."

"The Missouri Legislature passed a law last year restricting smoking in 'public places' to designated smoking areas."

"Illinois has an older smoking law, which also limits workplace smoking to designated areas."

"Both of these laws were passed prior to release of a recent Environmental Protection Agency report classifying secondary smoke or environmental tobacco smoke (ETS) as a known human carcinogen. . . . Because smokers comprise approximately 26 percent (or 50 million) of the adult United States population, *consuming a half trillion cigarettes annually*, exposure to ETS is nearly universal. The EPA's final report links ETS to lung cancer in adults and respiratory diseases, including asthma, in children."

"The Americans with Disabilities Act created some protection for individuals who are hypersensitive to tobacco smoke. . . . In 1991, the Occupational Safety and Health Administration began rulemaking procedures by issuing a public request for information on the general issue of indoor air pollution. Recently, OSHA announced that it would treat ETS separately from other indoor air pollutants, but has yet to take action on the ETS issue. OSHA also faces pressures from renewed lawsuits in an ongoing five-year battle with public interest groups that have been trying to force OSHA to ban workplace smoking."

"Carefully planned workplace smoking policies can offer some protection to employers. In fact, one out of every three American companies now bans or limits workplace smoking."

"A common courtesy approach, encouraging employees to resolve smoking issues among themselves, generally requires a great deal of flexibility and cooperativeness among the employees. . . . When successful, however, this type of policy is usually perceived as the most fair, primarily because of the greater degree of employment involvement."

"The ultimate goal of any smoking policy should be to accommodate all employees to the greatest extent possible in light of state and local requirements. . . . Whatever type of smoking policy is established, employers must ensure that the policy is well-planned and tailored to the particular employees and workplace at issue."

**[5] Marcus, S.E., Emont, S.L., Corcoran, R.D., Giovino, G.A., Pierce, J.P., Waller, M.N., and Davis, R.M., "Public Attitudes About Cigarette Smoking: Results from the 1990 Smoking Activity Volunteer Executed Survey," *Public Health Reports* 109: 125-134, 1994 [Issue 76, Item 42]**

Using data collected by American Cancer Society volunteers in a telephone survey, the authors report that 62-88 percent of respondents reported worksite smoking restrictions or nonsmoking areas, and that 26-48 percent reported being "bothered" by smoke at work.

EXCERPTS:

"The 1990 Smoking Activity Volunteer Executed Survey collected information on a wide range of policy-relevant issues concerning public attitudes about cigarette smoking. These issues include cigarette taxes, advertising restrictions, minors' access to tobacco products, school-based prevention, and exposure to environmental tobacco smoke in workplaces and public areas."

"Between 81 and 94 percent of the respondents reported that restaurants they patronize have nonsmoking areas most or all of the time. High percentages (between 81 and 87 percent) of former-never smokers ask to sit in these nonsmoking sections most or all of the time; between 19 and 21 percent of current smokers reported asking to sit in nonsmoking sections most or all of the time. Current smokers (between 46 and 59 percent) were less likely than were former-never smokers (between 74 and 78 percent) to think that the smoking ban on airlines has had a positive effect on people's health. More than half of former-never smokers reported ever asking someone not to smoke."

"Between 62 and 88 percent of respondents working outside the home in an enclosed building reported that the place where they work has nonsmoking areas or restrictions on smoking. Between 26 and 48 percent of working respondents, however, reported being bothered (in the past 12 months) by the amount of cigarette smoke at work. As expected, former-never smokers (between 38 and 55 percent) were more likely than were current smokers (between 11 and 21 percent) to have reported being bothered by environmental tobacco smoke. Moreover, only between 16 and 27 percent of former-never smokers reported ever complaining (in the past 12 months) to their supervisors about the amount of smoke at work."

"These data suggest that existing worksite tobacco control policies are not restrictive enough or are being inadequately enforced. The disparity between discomfort (being bothered by smoke at work) and behavior (com-

plaining to supervisors) suggests that it is difficult in work settings for people to speak out and voice their complaints rather than tolerate objectionable behavior."

"Because potential respondents and respondents knew that the person calling them was a volunteer from the American Cancer Society, it is possible that bias may have entered the study."

[6] **Pierce, J.P., Shanks, T.G., Pertschuk, M., Gilpin, E., Shopland, D., Johnson, M., and Bal, D., "Do Smoking Ordinances Protect Non-Smokers from Environmental Tobacco Smoke at Work?" *Tobacco Control* 3: 15-20, 1994 [Issue 78, Item 34]**

Based on data from the 1990-1991 California Tobacco Survey, the authors report a relationship between the strength of the local smoking ordinance and the degree of limitation of reported workplace ETS exposure. They claim that ETS exposure was likely to be less in workplaces with smoking restrictions if the local smoking ordinance was strong.

EXCERPTS:

"In this paper, we report population-based estimates of the percentages of California indoor workers covered by local smoking ordinances in 1990 and 1991. Further, we describe the association between the strength of these ordinances and the existence of worksite smoking policies and the exposure of non-smoking indoor workers to ETS."

"Non-smoker exposure to ETS appears to be lower in larger workplaces compared to smaller workplaces regardless of the strength of ordinance in effect. . . . In strong ordinance areas (regardless of workplace size) the overall percentage of non-smokers exposed to ETS was 24.5 ± 1.9%; in weak ordinance areas exposure was 29.1 ± 3.8%; and in no ordinance areas it was 34.8 ± 3.2%."

"[T]he level of ETS exposure is more strongly related to the strength of the worksite smoking policy than to the existence of an ordinance. Even in areas with no ordinance, exposure to ETS is low if the workplace is smoke-free. However, in worksites with a work area ban, but not a total ban, the existence of a strong ordinance appears to reduce the exposure to non-smokers to ETS, compared to areas with weak or no ordinances."

"Regions with workers reporting the highest percentages of smoke-free workplaces had the lowest levels of non-smoker ETS exposure."

"The current report establishes for the first time that strong local area smoking ordinances increase the likelihood that worksites have smoke-free policies. The variability among the 18 regions in California further demonstrates the relationship between increased non-smoker protection from ETS and the existence of strong smoking ordinances."

"In areas with strong local ordinances, work area smoking bans (a less restrictive category than a total ban) appear to be substantially more effective in reducing non-smoker exposure to ETS. One possible explanation for this finding is the right of the non-smoker covered by a strong ordinance to dictate workplace policy in the event of a conflict between a smoker and a non-smoker."

"Smoking ordinances can only be effective in protecting non-smokers from ETS if their provisions are actually implemented in the workplace. About 40% of the workers in our survey who worked in areas with strong ordinances reported minimal or no smoking restrictions in their workplace. This finding may reflect either a lack of knowledge on the part of the employees concerning existing smoking policies or the need for increased compliance with local ordinances."

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## STATISTICS AND RISK ASSESSMENT

[1] **Bero, L.A., Glantz, S.A., and Rennie, D., "Publication Bias and Public Health Policy on Environmental Tobacco Smoke," *Journal of the American Medical Association* 272: 133-136, 1994 [Issue 77, Item 42]**

Based on a comparison of articles published in symposia proceedings and journal articles, the authors claim that there is no publication bias against studies reporting statistically nonsignificant data on ETS. Referring to the *EPA Risk Assessment Litigation*, the authors suggest that negative studies should be carefully reviewed by the court, claiming that many are reviews that contain no statistical analysis.

EXCERPTS:

"This article tests the validity of the tobacco industry's argument that publication bias invalidates the EPA's risk assessment of ETS and other reviews of the health effects of ETS. . . . Since symposium proceedings on ETS comprise a substantial proportion of the literature on ETS, we assessed the extent of publication bias among articles on the effects of ETS on health that had been published as either peer-reviewed journal articles or proceedings of symposia."

"Proportions of statistically significant results and positive and negative conclusions were compared by . . . analysis of symposium vs peer-reviewed articles."

"In summary, we identified five unpublished negative studies, two of which were dissertations and none of which was cited among the 501 references in the final draft of the EPA risk assessment of ETS."

"Our results suggest that there is no publication bias against original research that does not report statistically significant results on the effects of ETS on health. Almost half of the original research published in the peer-reviewed literature reported statistically nonsignificant results or results of mixed significance. The lack of publication bias against negative studies on ETS may not correspond to the occurrence of publication bias as a whole because the great public interest in ETS might favor publication of negative results on ETS."

"Although similar proportions of both peer-reviewed journal articles and symposium articles on the health effects of ETS report statistically significant results, a larger proportion of journal articles than symposium articles have negative conclusions, ie, conclusions that ETS exposure is not harmful. Although 80% of the journal articles had positive conclusions, only 57% of the studies actually reported statistically significant results. This discrepancy is primarily caused by the reporting of results of mixed significance as being positive. In contrast, the symposia report results of mixed significance as being negative. When health outcomes are studied, drawing positive conclusions from results of mixed significance is justified because an intervention (such as ETS exposure) can have adverse health consequences when it affects one physiologic measure, even if it does not affect every possible physiologic measure associated with the health outcome."

"In the symposia, the large proportion of articles with negative conclusions primarily results from the inclusion of 22 review articles and 19 original articles that did not report statistical tests. . . . As none of the review articles identified in this study reported the methods used for pooling data or performing statistical tests, it was impossible to evaluate the selection criteria for articles or the validity of the conclusions."

"The finding that the symposia contain more review articles and articles without statistical analyses concluding that ETS is not harmful than do peer-reviewed journals supports the supposition that sponsorship can influence the presentation of results as being positive or negative. . . . In the ETS symposia, there appears to be a bias toward the publication of negative conclusions that are not supported by the available data."

"The tobacco industry has used the possibility of publication bias to argue that reviews of the literature on the health effects of ETS are invalid. In the case of *Flue-Cured Tobacco Cooperative Stabilization Corp v EPA*, the tobacco industry claims that publication bias invalidates the EPA risk assessment because negative studies have been excluded. Our findings suggest that negative studies, such as those published in symposia, should be carefully scrutinized before they are included in reviews or considered by the courts because the negative conclusions may not be supported by any statistical analyses. . . . For negative unpublished data, we recommend that the courts, as well as all scientists, assess whether the data have been submitted for publication in a peer-reviewed journal, whether the article presents original research, whether the article actually reports statistical tests, the quality of the methods, and the source of funding for the research."

**[2] Sullum, J., "Passive Reporting on Passive Smoke," *MediaCritic* 41-47, 1994 [Issue 76, Item 43]**

The author of this article uses press coverage of the EPA Risk Assessment on ETS to discuss his position that journalists should exercise skepticism and cover both sides of issues such as ETS equally. The article was recently reprinted as part of a Philip Morris advertisement that ran in major newspapers on July 3, 1994.

EXCERPTS:

"In light of the legislation and policy changes it has generated, the EPA's *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders* may be the most influential report ever issued by the agency. As one might expect, it has received extensive coverage from major newspapers. . . . Yet almost without exception, the coverage has been one-sided, credulous and superficial. . . . And despite serious questions about the report's assertion that ETS causes lung cancer and the process by which the EPA reached that conclusion, leading U.S. newspapers have treated this assertion as scientific fact. In so doing, not only have they exaggerated what is known about the effects of ETS, but they have missed an important story about the corruption of science by the political crusade against smoking."

"Faced with evidence that was weak, inconsistent and ambiguous, the EPA finessed some important points and gave the data a vigorous massage to arrive at the conclusion that ETS causes lung cancer. To begin with, the EPA used an unconventional definition of statistical significance."

"The contrivances employed by the EPA, which a July 31, 1992 *Science* article described as 'fancy statistical footwork,' indicate that the agency was determined to reach the conclusion that ETS kills non-smokers. That impression is supported by the fact that the EPA put together a 'policy guide' for reducing workplace exposure to ETS well before it had officially decided that ETS was a hazard."

"Despite these and other warning signs, the coverage by the major newspapers was generally unskeptical of the agency's conclusions and dismissive of the tobacco industry's criticism. The typical story opened with the government's claims, elaborated on them for several paragraphs, quoted anti-smoking activists who agreed with the EPA and described the tobacco industry's response in a paragraph or two. The tobacco industry's comments usually amounted to little more than a denial, and no independent sources were provided to back them up. News consumers were left with the impression that, aside from industry representatives, no one had doubts about the EPA's position on the health effects of ETS."

"[E]rrors in stories about the EPA report reflect a general tendency in coverage of the ETS controversy to exaggerate evidence and minimize criticism."

"Another common error involves confusing correlation with causation."

"The errors that appear in these and other stories about ETS are not random, of course. They consistently weigh in favor of the view that ETS is a serious health hazard. Reporters are receptive to that view for a number of reasons. Even if they don't personally disapprove of smoking, they are well aware of its dangers. If a lot of tobacco smoke hurts smokers, it seems plausible that a little would hurt non-smokers, though not as much."

"Since most journalists do not have backgrounds in statistics or epidemiology, they rely on other people to assess the issue. The most conspicuous sources for stories about ETS work for the tobacco industry, the government and anti-smoking groups. Reporters don't trust the tobacco companies. But in contrast to the skepticism they bring to the pronouncements of other government agencies and special-interest groups, they do tend to trust public-health authorities such as the EPA and anti-smoking organizations such as the American Cancer Society. The governing assumption seems to be that the tobacco companies are trying to maintain profits, while the government and anti-smoking groups are interested in promoting public health and getting out the facts."

"But sometimes these two missions conflict. Public health officials may be inclined to shade the truth a bit if it helps to discourage smoking by making it less acceptable."

"When reporters choose sides on the basis of trust, they fail to make independent assessments of the arguments of both sides. So readers of stories about ETS might wish to keep in mind the following points:"

"The Importance of Statistical Significance. When researchers do not come up with statistically significant results, they tend to underplay this fact, for obvious reasons. Stories should be examined to see whether they disclose, as a good report should, whether a result is statistically significant."

"The Pitfalls of Correlation versus Causation. Even a statistically significant association between A and B does not prove that A causes B. A and B could both be associated with another factor or set of factors."

"The concern about confounding variables is especially important when risk ratios are small."

"Weasel Words. Readers should be alert to qualifiers and hedging; so should reporters. . . . When someone cites a 'pattern' or a 'trend in the data,' it's time to look more closely. In rigorous science, close doesn't count."

"Discrepancies. When two versions of a verifiable fact diverge sharply, readers should reserve judgment."

"If reporters want to get at the truth, they cannot continue to act as if only one side in this debate has an ax to grind. They need to be just as skeptical about the EPA and the Coalition on Smoking or Health as they are about Philip Morris."

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